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Electrogenic events associated with electron and proton transfers within the cytochrome b_6/f complex

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Abstract

The kinetics and amplitude of the membrane potential changes associated with electron and proton transfers within the cytochrome b_6/f (cyt *b*/*f*) complex (phase b) are measured in vivo in *Chlamydomonas reinhardtii* under anaerobic conditions. Upon saturating flash excitation, fast components in the membrane potential decay superimposed on phase b lead to an underestimation of the amplitude of this phase. In the FUD50 mutant strain, which lacks the ATP synthase, the decay of the membrane potential is slowed down compared to the wild type, and the kinetics and amplitude of phase b may be accurately determined. This amplitude corresponds to the transfer of at least 1.5 charges across the membrane per positive charge transferred to photosystem I, whatever the flash energy. This value largely exceeds that predicted by a Q-cycle process. Similar conclusions are reached using the wild type strain in the presence of 9 μM dicyclohexylcarbodiimide, which specifically inhibits the ATP synthase. It is concluded that a proton pumping process is operating in parallel with the Q-cycle, with a yield of ∼0.5 proton pumped by cyt *b*/*f* complex turnover, irrespective of the flash energy.

Keywords: Cytochrome *b*/*f* complex; Electrogenic reaction; Electron transfer; Proton pump

1. Introduction

The cytochrome b_6/f (cyt *b*/*f*) complex is a membrane protein that catalyzes the electron transfer between plastoquinol and plastocyanin in the oxygen evolving apparatus. The cyt *b*/*f* complex includes four electron carriers. Cyt *f* and the Rieske iron sulfur protein (FeS) form the high potential chain and are located close to the lumen. The cyt *b* hemes, cyt b_l (low potential cyt *b*) and cyt b_h (high potential cyt *b*) are in transmembrane positions and located close to the lumen and stroma, respectively (reviewed in [1]). Plastoquinol is oxidized at the Q_o site close to the lumen while plastoquinone is reduced at the Q_i site close to the stroma. A fifth electron carrier, G, [2,3] located on the stromal face of the cyt *b*/*f* complex shares one electron with cyt b_h.

A membrane potential increase is associated with the turnover of the cyt *b*/*f* complex. The variation of the membrane potential is measured by the electrochromic shift of membrane pigments, which induces large absorption changes around 518 nm [4]. Upon illumination by a short flash, one observes a fast increase (phase a, <100 μs) of the membrane poten-

Abbreviations: cyt *b*/*f*, cytochrome b_6/f; cyt b_h, high potential cytochrome *b*; cyt b_l, low potential cytochrome *b*; DCCD, *N*,*N*′-dicyclohexylcarbodiimide; FCCP, carbonyl cyanide 4-(trifluoromethoxy)phenylhydrazone; FeS, Rieske iron sulfur protein; PS, photosystem

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tial proportional to the number of charge separations occurring at the level of reaction centers [4]. Phase a is followed by a slower phase (phase b) completed in less than 200 ms, associated with the transmembrane movement of charges within the cyt *b/f* complex.

The processes of electron transfer and proton pumping within the cyt *b/f* complex are generally interpreted in the framework of the Q-cycle (Scheme 1), initially proposed by Mitchell [5] and modified by Crofts et al. [6]. In whole algae under anaerobic conditions, the plastoquinone pool is fully reduced and the illumination induces an efficient cyclic electron flow which only involves the cyt *b/f* complex and photosystem 1 (PS 1). Under these conditions, the electron transfer process within the cyt *b/f* complex is initiated by the transfer of a positive charge from P_{700} to the high potential chain via the diffusion of oxidized plastocyanin. The sequence of electron transfer reactions depends upon the state of the *b* hemes prior to the illumination, reactions 1 to 3 from state ($b_l^+ b_h^+$) or reactions 1′ to 3′ from state ($b_l^+ b_h$). A complete Q-cycle requires the transfer of two positive charges to the high potential chain, which induces the oxidation of two plastoquinols at site Q_o (reactions 2 and 2′) and the reduction of one plastoquinone at site Q_i (reaction 3′). Only reactions 3 and 3′ are electrogenic, i.e. associated with a transmembrane movement of electrons or protons [7]. Assuming similar structures for cyt *b/f* and cyt b/c_1 of purple bacteria, the electrogenic phase associated with the reduction of cyt b_h^+ (reaction 3) is ~ 0.6 of that corresponding to the transfer of one electron from the lumen to the stroma [7]. The electrogenic phase associated with the transfer of one electron from cyt b_l to cyt b_h and two protons from the stroma to site Q_i is $\sim 0.6 + 2 \times \sim 0.4 = \sim 1.4$ (reaction 3′). Therefore, upon a complete Q-cycle, the total amplitude of phase b is $\sim 0.6 + \sim 1.4 = \sim 2$ for two electrons transferred to the high potential chain. Such a stoichiometry is characteristic of all redox-loop mechanisms initially proposed by Mitchell [8]. These mechanisms exclude the involvement of proton channels spanning the whole membrane thickness. The addition of specific inhibitors, or alternatively mutations of the polypeptides in the cyt *b/f* complex, can then slow down the rate of electron transfer and electrogenic reactions with no alteration of the proton to electron stoichiometry.

Under anaerobic conditions, a fermentation process continuously synthesizes ATP, the hydrolysis of which induces a permanent electrochemical proton gradient [9]. This gradient [10], and more specifically its osmotic component [11], slows down the rate of electron transfer reactions within the cyt *b/f* complex. The addition of uncouplers as carbonyl cyanide 4-(trifluoromethoxy)phenylhydrazone (FCCP) [10] or nigericin [11] accelerates the electron transfer and electrogenic reactions.

The following observations cannot be interpreted on the sole basis of the Q-cycle and suggest the involvement of an additional proton pumping process.

1. When both *b* cytochromes are reduced, i.e. in the presence of sodium dithionite and a mediator, the concerted process (Scheme 1, reactions 2 and 2′) cannot operate. Under these conditions, one observes a flash induced oxidation of cyt b_l associated with the reduction of the high potential chain [10,12,13], as follows:

$$PQH_2 + (\text{FeS cyt } f)^+ \rightarrow PQ^- + (\text{FeS cyt } f) + 2H^+ \quad \text{(a)}$$

$$PQ^- + \text{cyt } b_l + 2H^+ \rightarrow PQH_2 + \text{cyt } b_l^+ \quad \text{(b)}$$

   We have observed in algae that Reaction b is associated with an electrogenic phase of amplitude close to 1 [10,13]. We have proposed [13] that at least one of the two protons associated with Reaction b is taken up from the stroma via a transmembrane proton channel, by a mechanism similar to that proposed by Wikström and Krab [14] for cyt oxidase.
2. According to Scheme 1, the illumination of a cyt *b/f* complex in the ($b_l^+ b_h$) state leads to a sequential process of two reactions: reaction (2′), associated with the reduction of cyt b_l^+ is non-electrogenic while reaction (3′), associated with the reduction of plastoquinone, induces the transfer of ~ 1.4 charges across the membrane. Actually, we do not observe in the kinetics of phase b, the lag phase predicted by the sequential occurrence of reactions (2′) and (3′). The ratio of the initial rate of phase b to the rate of cyt b_l^+ reduction corresponds to the transmembrane transfer of about one charge per cyt b_l^+ reduced [10]. This

result suggests that a transmembrane movement of one proton is coupled to the oxidation of plastoquinol at site Q_o [15].

3. In the presence of *N*,*N*′-dicyclohexylcarbodiimide (DCCD), the kinetics of phase b displays a lag phase close to that predicted on the basis of a Q-cycle process and we had therefore proposed that DCCD specifically inhibits the additional proton pumping process [15]. At a higher concentration of DCCD, we observed, in agreement with Wang and Beattie [16], a partial uncoupling of the proton pumping process from the electron transfer. A lag in phase b is similarly observed after H_2O/D_2O substitution [17].
4. Under subsaturating flash excitation given to whole algae under anaerobic conditions, the amplitude of phase b is larger than that predicted from a Q-cycle process. Upon a very weak-flash illumination (hitting ∼3% of the PS 1 centers), we have observed in *Chlorella sorokiniana* (S8 strain) phase b of ∼1.8 in amplitude [15]. Upon excitation by saturating flashes, the amplitude of phase b is somewhat variable from one batch to the other but remains close to that predicted for a Q-cycle process. Most of the arguments that favor the occurrence of an additional proton pumping process are based on the measurement of the amplitude and kinetics of phase b, that are subjected to various sources of error. First, in the spectral range chosen to measure the electrochromic shift, additional absorption changes irrelevant to the membrane potential changes may occur. Second, the kinetics of phase b must be corrected for the decay of the membrane potential associated with ionic leaks through the membrane or via the ATPase F_0 channel. We used a deconvolution procedure (see Section 2) based on the assumption that the rate of the ionic leaks is proportional to the membrane potential value (first-order reaction). Should this assumption prove to be incorrect, the deconvolution procedure would lead to an underestimation of phase b.

In this paper, we propose a reliable method to determine the amplitude of the electrogenic phase associated with electron and proton transfers within the cyt *b*/*f* complex. On this basis, the involvement of a proton pumping process superimposed to the Q-cycle process is addressed.

2. Materials and methods

Experiments were performed using *Chlamydomonas reinhardtii* wild type (WT) and two mutant strains, Δ*petD* [18] and FUD50 [19]. The Δ*petD* strain lacks subunit IV and therefore does not assemble the cyt *b*/*f* complex. The FUD50 strain is deleted in the *atpB* gene encoding subunit β of CF_1 [20].

Algae were suspended in 20 mM HEPES buffer, pH 7.2, 20% (w/v) Ficoll. The experiments were performed under anaerobic conditions in the presence of 0.1 mM hydroxylamine and 10 μM 3-(3′,4′-dichlorophenyl)-1,1-dimethylurea. Stock solutions of DCCD were prepared in dimethylsulfoxide. DCCD at a given concentration was added to the algae in growth medium and incubated for 40 min. Then after centrifugation, the pellet was suspended in the buffer containing DCCD at the same concentration as used during the incubation.

Spectrophotometric measurements were performed with an apparatus similar to that described in [21,22]. Actinic excitation is provided by xenon flashes (3 μs half-time duration) filtered through RG8 Schott red filters. Since the flash duration is of the same order of magnitude as P_{700}^{+} reduction, the highest energy actinic flashes induce ∼1.3 PS 1 turnovers (oversaturating flashes). The variation of the delocalized membrane potential has been measured by the electrochromic shift of membrane pigments as the difference (518–548 nm). In this difference, and in the 100 μs–200 ms range of time, the negative signal associated with the reduction of plastocyanin (∼2% of the amplitude of phase a) is about equal to the positive signal associated with the reduction of cyt *f*. We therefore neglect the spectral changes associated with plastocyanin and cyt *f*. We have checked also that the difference (518–548 nm) is linearly related to the value of the membrane potential.

The kinetics of phase b are computed from the kinetics of the membrane potential change after correction for its decay which is associated with ion leaks through the membrane or via the ATPase. The deconvolution procedure is based on the assumption that the rate of the membrane potential

decay is proportional to its amplitude (first-order process). The rate constant of the decay is determined after completion of phase b (>200 ms after the actinic flash). We discuss here the validity of this deconvolution procedure under different experimental conditions.

3. Results and discussion

3.1. ΔpetD mutant strain lacking of cyt b/f complex

Fig. 1 shows the kinetics of absorption changes measured at 518 nm (curve 1) and 548 nm (curve 2) induced by an oversaturating flash. Since PS 2 reaction centers are inhibited, the absorption increase observed at both wavelengths in the range of 0–200 ms is likely due to the electron transfer reactions occurring at the level of PS 1 acceptors. Computing the difference 518–548 nm (curve 3) cancels this increasing phase. After correction for the decay of the membrane potential, using the deconvolution procedure described in Section 2, the difference (518–548 nm) is close to a step function, displaying no significant slow membrane potential increase, as expected for a mutant lacking the cyt *b/f* complex (curve 3′). We thus conclude that the signal measured as the difference (518–548 nm) is a reliable indicator of the membrane potential changes. In the presence of 0.3 μM FCCP, the membrane potential decay is no longer exponential but displays fast components completed in ∼200 ms (not shown).

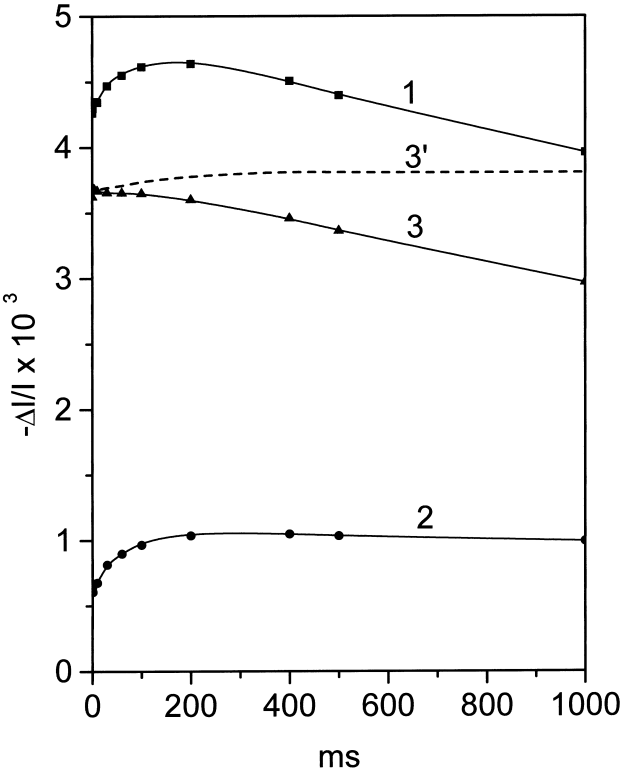


Fig. 1. Kinetics of absorption changes. *C. reinhardtii* Δ*petD* mutant strain. Saturating actinic excitation. Curve 1, 518 nm; curve 2, 548 nm; curve 3, (518–548 nm); curve 3′, deconvolution of curve 3, assuming an exponential membrane potential decay. The rate constant of the decay is measured in the 500 ms–1 s time interval.

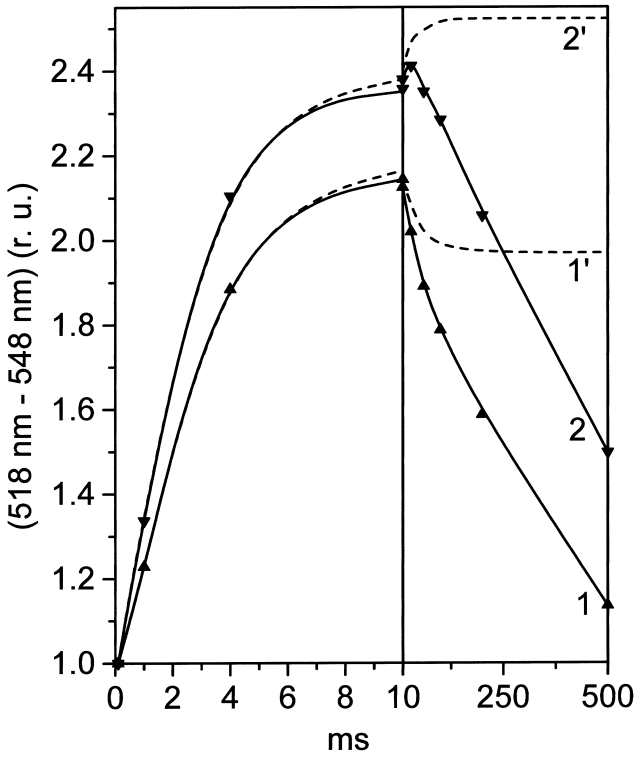


Fig. 2. Kinetics of the membrane potential changes (expressed as the difference (518–548 nm) and normalized to the amplitude of phase a) at two energies of the actinic flash. *C. reinhardtii* WT. 0.5 μM FCCP. Saturating flash, curve 1. Subsaturating flash, hitting ∼15% of PS 1 reaction centers, curve 2. Curves 1′ and 2′, same as curves 1 and 2, respectively, after correction for the membrane potential decay. The rate constant of the membrane potential decay is measured in the 200–500 ms time interval.

3.2. Wild type

Fig. 2 shows the kinetics of phase b measured in

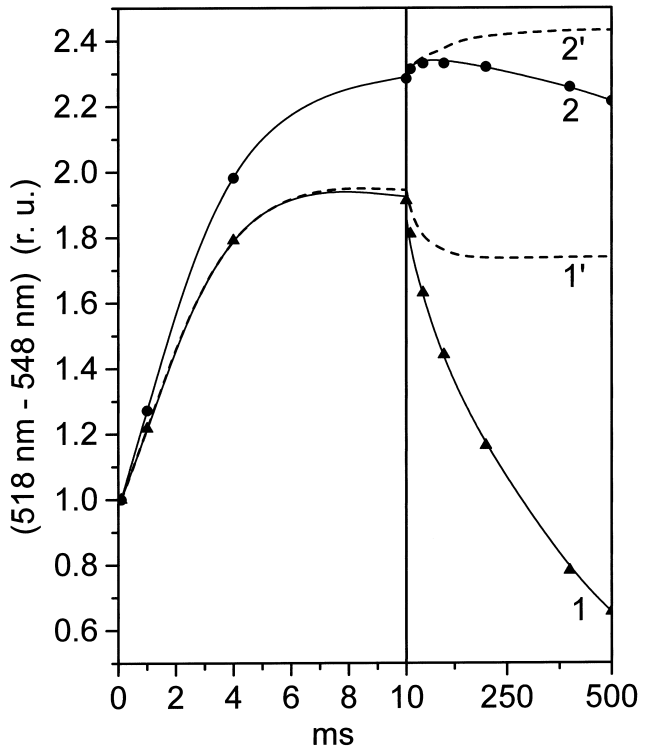


Fig. 3. Kinetics of the membrane potential changes (expressed as the difference (518–548 nm) and normalized to the amplitude of phase a). *C. reinhardtii* WT. Saturating actinic excitation. Curve 1, 0.9 μM FCCP. Curve 2, 9 μM DCCD. Curves 1′ and 2′, same as curves 1 and 2, respectively, after correction for the membrane potential decay. The rate constant of the membrane potential decay is measured in the 200–400 ms time interval for curve 1′ and in the 500 ms–1 s time interval for curve 2′.

the presence of 0.5 μM FCCP added to collapse the permanent membrane potential induced by the hydrolysis of ATP. Curves 1 and 2 are measured following an oversaturating-flash or a subsaturating flash excitation hitting ∼15% of PS 1 centers, respectively. Curves 1′ and 2′ are computed from curves 1 and 2 after deconvolution for the decay of the membrane potential. Curve 1′ displays a decaying component completed in ∼200 ms, which shows that the assumption of an exponential decay of the membrane potential used in the deconvolution procedure is not satisfied. As reported above, a similar behavior is observed with Δ*petD* mutant in the presence of FCCP. Fast components in the membrane potential decay superimposed on phase b make the measurement of its amplitude unreliable and led to its underestimation. Under a subsaturating flash excitation, the membrane potential decay is closer to an exponential function, which permits a more reliable estimation of the amplitude of phase b (curve 2′).

Fig. 3 shows the kinetics of the membrane potential changes computed before (solid curves) or after (dashed curves) deconvolution for the membrane potential decay in the presence of either 0.9 μM FCCP (curves 1 and 1′) or 9 μM DCCD (curves 2 and 2′), after an oversaturating flash excitation. As already stated, in the presence of FCCP, the decay of the membrane potential is non-exponential.

As expected, the addition of 9 μM DCCD induces a slowdown of the membrane potential decay ($t_{1/2}$ ∼5 s), owing to the inhibition of the membrane ATPase; this very slow decay allows an accurate measurement of the amplitude of phase b (1.42). The initial rate of phase b is ∼20% larger in the presence of 9 μM DCCD than in the presence of 0.9 μM FCCP. This suggests that during the first ms following the flash ∼20% of the protons released in the lumen by the cyt *b/f* complex are back-transferred to the stroma via the uncoupler FCCP or via

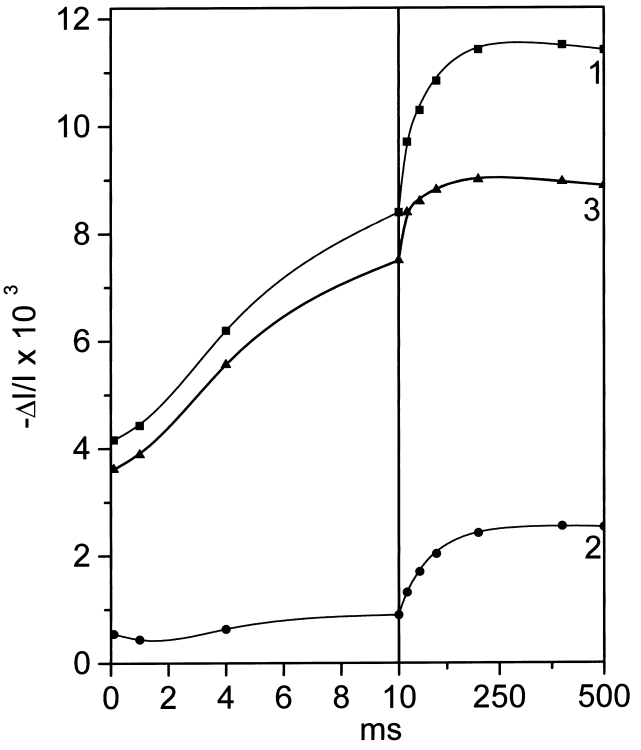


Fig. 4. Kinetics of absorption changes. *C. reinhardtii* FUD50 mutant strain. Oversaturating flash excitation. Curve 1, 518 nm; curve 2, 548 nm; curve 3, (518–548 nm).

the F_0 channel. It is worth noting that DCCD at a higher concentration (>20 μM) induces opposite effects, i.e. a lag in the kinetics of phase b associated with a decrease of its amplitude [15]. The experiments (Fig. 3) show that the amplitude of phase b is largely underestimated in the presence of FCCP and active F_0F_1 ATPase.

3.3. *FUD50 mutant strain lacking the F_0F_1 membrane ATPase*

Fig. 4 shows the absorption changes measured at 518 nm (curve 1), 548 nm (curve 2) and in the difference (518–548 nm) (curve 3) induced by an oversaturating flash. An absorption increase completed in about 200 ms is observed at 548 nm, a wavelength where the contribution of the electrochromic shift is small. This increase is clearly of different origin than that we have observed at the same wavelength in the Δ*petD* mutant, since its amplitude is 3–4 times larger in the FUD50 mutant for the same PS 1 reaction centers concentration. We measured this signal in the 30–200 ms time-interval at 489, 548 and 573 nm, i.e. at wavelengths where the contribution of the membrane potential is small. The amplitude is the same at the three wavelengths within 5%. We tentatively ascribed this absorption increase to a flash-induced light-scattering associated with a swelling of the thylakoid, which might be due to a proton release in the lumen. Assuming that this absorption increase has a flat spectrum in this spectral range, its contribution is cancelled by computation of the difference (518–548 nm) which then becomes a reliable determination of the membrane potential change.

Fig. 5 shows the kinetics of phase b, corrected for the membrane potential decay. In the control, the amplitude of phase b is 1.58 under an oversaturating flash excitation (curve 1) and 1.72 under a subsaturating flash excitation (curve 2). The addition of 0.5 μM nigericin, a proton/K^+ antiporter, induces an acceleration of phase b of ∼20% with no significant change of its amplitude (curve 3). The addition of 0.3 μM FCCP induces a similar acceleration of phase b, but decreases its amplitude ∼12% (not shown). In the presence of 9 μM DCCD (curve 4), the initial rate of phase b is equal to that measured in the presence of nigericin, with a ∼10% decrease in its amplitude. This suggests that a concentration of

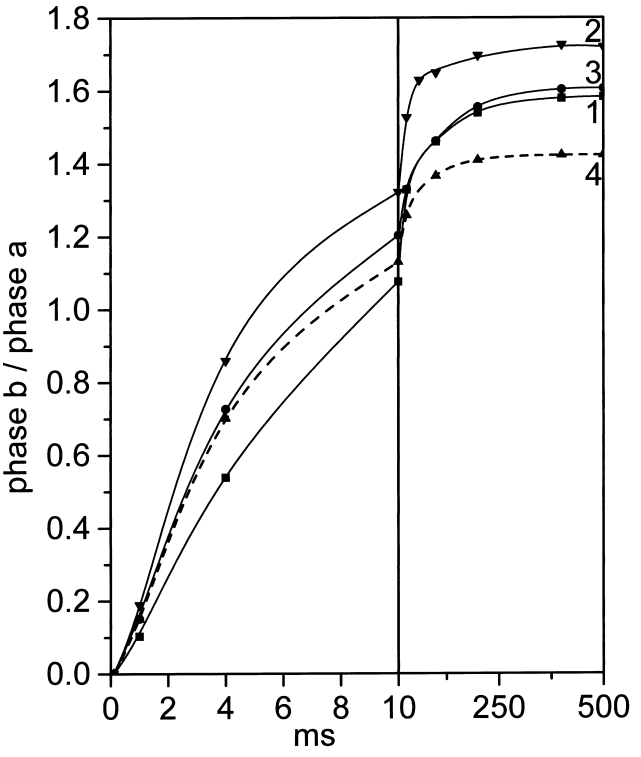
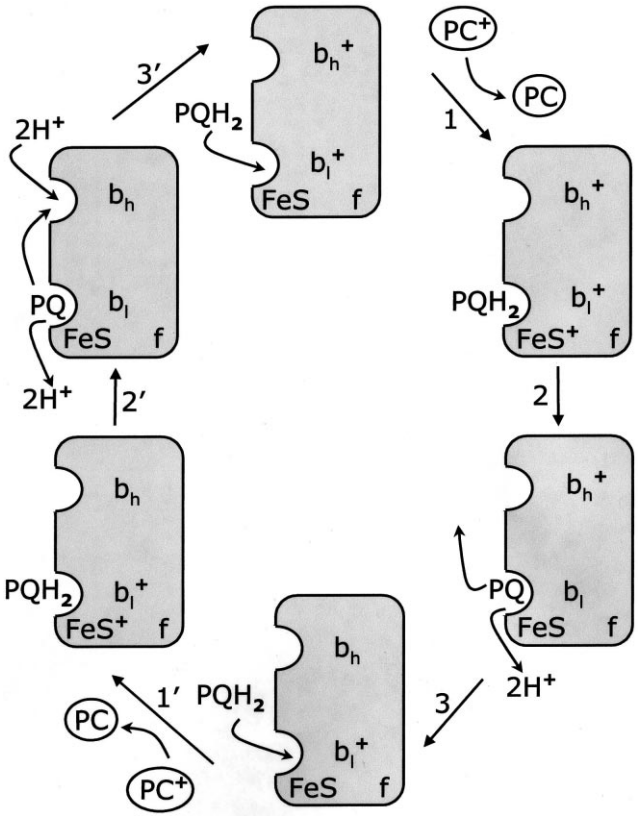


Fig. 5. Kinetics of phase b corrected for an exponential membrane potential decay. *C. reinhardtii* FUD50 mutant strain. Phase b is normalized to phase a. The rate constant of the decay is measured in the 500 ms–1 s time interval. Saturating flash excitation: curve 1, control; curve 3, 0.5 μM nigericin; curve 4 (dashed), 9 μM DCCD. Subsaturating flash excitation, hitting ∼20% of PS 1 reaction centers: curve 2, control.

9 μM DCCD is sufficient to induce a slight inhibition of proton channels within the cyt *b/f* complex. The amplitude (1.41) is about equal to that measured in the WT under the same conditions (Fig. 3, curve 2′), showing that DCCD has similar effects on the cyt *b/f* complex in both the WT and FUD50 strains.

The effect of nigericin suggests that in FUD50 strain an unknown proton pump establishes a permanent proton gradient collapsed by this uncoupler. We can exclude the presence of traces of F_0F_1 ATPase since the FUD50 mutant strain is obtained by deletion of the gene encoding the β subunit of the F_1 factor. A similar effect of nigericin is observed in ac 46 mutant strain that lacks the F_0 channel (not shown). The fact that DCCD induces a stimulation of the initial rate of phase b similar to that observed in the presence of nigericin implies that this putative proton pump is inhibited by DCCD. We therefore propose that this pump involves a proton channel



Scheme 1.

including dicarboxylic acids able to bind DCCD. Rappaport et al. [23] have previously reported that in SL8 strain of *C. sorokiniana* lacking the F_0F_1 ATPase, an unknown ATP-driven ionic pump was responsible for a large permanent membrane potential. At variance with us, these authors do not observe any significant acceleration of phase b induced by nigericin, which excludes the involvement of a proton pump. We cannot exclude that the ionic pump mentioned by Rappaport et al. [23] is also present in the FUD50 strain.

The amplitude of phase b in the FUD50 strain, which is determined precisely owing to the slow decay of the membrane potential, is higher than that predicted from a simple Q-cycle process, irrespective of the flash excitation energy. We previously established [10] that after several minutes of dark anaerobic adaptation, cyt b_h is fully reduced and most of the cyt *b/f* complexes are in the (cyt b_l^+ cyt b_h) state. Upon illumination by a weak flash, the probability for a cyt *b/f* complex to undergo double turnovers is small and, according to a Q-cycle process, reactions 1′ to 3′ (Scheme 1) lead to the oxidation of cyt b_h and to a phase b of amplitude $\sim$1.4, a value very likely overestimated since the positive charges are shared between cyt b_h and the additional carrier G, located on the stroma side of the membrane [3]. We conclude that under a weak-flash excitation, the measured amplitude of phase b is 0.4 to 0.5 larger than that predicted by a Q-cycle process. This result is similar to that obtained in the S8 mutant strain of *C. sorokiniana* upon excitation by very weak flashes and was interpreted by assuming that the additional proton pumping process is coupled to reaction 2′ in Scheme 1 [15].

After an oversaturating flash which induces $\sim$1.3 PS 1 charge separations and taking into account that the stoichiometry cyt *f*/P_{700} is $\sim$0.7 [10], a major fraction of the cyt *b/f* complexes undergoes double turnovers. For these complexes, which undergo a complete Q-cycle, one expects, whatever the initial redox state of cyt b_h, an amplitude of phase b equal to 1 and no net oxidation or reduction of cyt b_h. In the experiment (Fig. 5), on the basis of the amount of cyt b_h oxidized by an oversaturating flash we estimate that $\sim$90% of the cyt *b/f* complexes undergo double turnover. The amplitude of phase b measured in FUD50 strain ($\sim$1.6) is therefore $\sim$0.5 larger than that predicted from a Q-cycle process. We therefore conclude that an additional proton pumping process operates with a similar yield after weak flashes and after an oversaturating flash i.e. when the proton pump is operating against a large membrane potential. We have previously proposed that the formation of a negatively charged semiquinone at site Q_o induces the transmembrane pumping of one proton driven by electrostatic interaction [15]. Proton pumping can involve a p*K* shift of a dicarboxylic amino acid localized in the vicinity of site Q_o [15,24] or a direct protonation of the semiquinone itself [25]. On the basis of the results presented here we propose that, at variance with the model discussed in [15], the proton pumping process is coupled as well to reaction 2 as to reaction 2′ (Scheme 1). Under anaerobic conditions, the yield is $\sim$0.5 proton pumped per cyt *b/f* complex turnover, which implies that about half of the pumped protons goes back to the stroma side of the membrane and half is released in the lumen via the transmembrane pro-

ton channel. The efficiency of this intracomplex proton leak will modulate the amplitude of phase b.

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References

[1] W.A. Cramer, G.M. Soriano, M. Ponomarev, D. Huang, H. Zhang, S.E. Martinez, J.L. Smith, Annu. Rev. Plant Physiol. Plant Mol. Biol. 47 (1996) 477–508.
[2] J. Lavergne, Biochim. Biophys. Acta 725 (1983) 25–33.
[3] P. Joliot, A. Joliot, Biochim. Biophys. Acta 933 (1988) 319–333.
[4] W. Junge, H.T. Witt, Z. Naturforsch. 23b (1968) 244–254.
[5] P. Mitchell, FEBS Lett. 57 (1975) 137–139.
[6] A.R. Crofts, S.W. Meinhardt, K.R. Jones, M. Snozzi, Biochim. Biophys. Acta 723 (1983) 202–218.
[7] D.E. Robertson, P.L. Dutton, Biochim. Biophys. Acta 935 (1988) 273–291.
[8] P. Mitchell, Nature 191 (1961) 144–148.
[9] P. Joliot, A. Joliot, Biochim. Biophys. Acta 975 (1989) 355–360.
[10] P. Joliot, A. Joliot, Proc. Natl. Acad. Sci. USA 91 (1994) 1034–1038.
[11] G. Finazzi, F. Rappaport, Biochemistry 37 (1998) 9999–10005.
[12] M.E. Girvin, W.A. Cramer, Biochim. Biophys. Acta 767 (1984) 29–38.
[13] P. Joliot, A. Joliot, Biochim. Biophys. Acta 849 (1986) 211–222.
[14] M. Wikström, K. Krab, Biochim. Biophys. Acta 549 (1979) 177–222.
[15] P. Joliot, A. Joliot, Biochemistry 37 (1998) 10404–10410.
[16] Y. Wang, D.S. Beattie, Arch. Biochem. Biophys. 291 (1991) 363–370.
[17] C. Deniau, F. Rappaport, Biochemistry 39 (2000) 3304–3310.
[18] R. Kuras, F.-A. Wollman, EMBO J. 5 (1994) 1019–1027.
[19] P. Bennoun, Proc. Natl. Acad. Sci. USA 79 (1982) 4352–4356.
[20] J.P. Woessner, A. Masson, E.H. Harris, P. Bennoun, N.W. Gillham, J.E. Boyton, Plant Mol. Biol. 3 (1984) 177–190.
[21] P. Joliot, D. Béal, B. Frilley, J. Chem. Phys. 777 (1980) 209–216.
[22] P. Joliot, A. Joliot, Biochim. Biophys. Acta 765 (1984) 210–218.
[23] F. Rappaport, G. Finazzi, Y. Pierre, P. Bennoun, Biochemistry 38 (1999) 2040–2047.
[24] U. Brandt, B. Trumpower, Crit. Rev. Biochem. Mol. Biol. 29 (1994) 165–197.
[25] A. Joliot, P. Joliot, in: G. Garab (Ed.), Photosynthesis: Mechanisms and Effects, vol. III, Kluwer Academic, Dordrecht, 1998, pp. 1573–1576.